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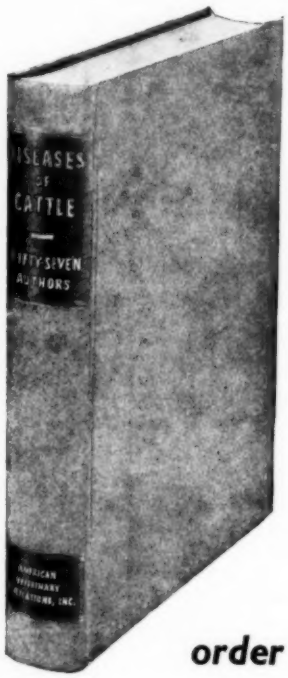

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Some Observations on Infectious Laryngotracheitis

BY

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SUMMARY.—The symptoms, epidemiology, lesions and diagnosis of infectious laryngotracheitis in 11 outbreaks in Lancashire and Cheshire are described.

The symptoms comprised coughing, sneezing, râles, gasping respiration, panophthalmia and nasal exudate. Expectoration of blood was not seen. Morbidity was high and mortality ranged from 0 to 50 per cent.

The disease affected birds varying in age from 5 weeks to 2 years, of a variety of breeds, and kept under all the usual types of management. The purchase or retention of survivor birds which were carriers was the commonest source of infection. Spread from pen to pen was often irregular and slow.

The most constant lesions were caseous and mucous exudates in the trachea and bronchi. Blood clots in the trachea were only rarely found.

The type of disease seen in these outbreaks cannot be readily distinguished from other respiratory diseases, including the respiratory form of Newcastle disease, except by the detection of the virus in the laboratory, the technique for which has briefly been described. The best material for isolation of the virus is obtained from swabs from the trachea and ocular discharge in living birds and from the trachea and lungs post-mortem. It is difficult to detect virus in birds which have shown symptoms for longer than a week.

Possible methods of control of the disease have been discussed.

INFECTIOUS largyngotracheitis of poultry is a disease affecting the respiratory tract in which lesions and exudate are constantly found in the trachea and larynx, and less frequently in the other parts of the respiratory system. The eyes are often affected.

The disease was first reported by May and Titsler (1925), who described an outbreak in fowls on Rhode Island, U.S.A., in 1923. They showed it to be readily transmissible. Beaudette (1930) first showed that the disease was caused by a filtrable virus.

The existence of the disease in Britain was first recorded by Dobson in 1935 (Dobson 1935a, b) and since then there have been no further published references to the disease in this country except for the papers by Chu (1954, 1958).

This paper confirms the presence of the disease in England, and records the clinical and epidemiological findings in 11 outbreaks on 10 farms, 6 in Lancashire and 4 in Cheshire. Visits were not made to these farms until the possible existence of Newcastle disease had been eliminated by the Animal Health branch of the Ministry of Agriculture, Fisheries and Food, and in fact 8 of them were initially reported as suspected fowl pest.

The period of observation on the farms varied between 6 and 15 months. At the end of 6 months the stock on 4 of the farms in Lancashire had been slaughtered because of fowl pest.

The farms ranged in size from one carrying 400 birds to a farm of 14,000. During the clinical manifestation of the disease frequent visits were made to affected farms, followed later by visits at intervals of about a month. Each outbreak was confirmed as infectious laryngotracheitis by the identification of the virus from affected birds by the production of typical pocks on the chorioallantoic membrane of the chick embryo, and these pocks were differentiated from those caused by the virus of fowl pox by scarification of pock material into the comb of susceptible fowl.

Clinical character of the disease

The disease was seen in fowls from 5 weeks to 2½ years of age, in both sexes, and in a variety of breeds and crossbreeds. There was no evidence of an age resistance. All affected fowls showed depression and malaise which varied in their manifestation from a slight reduction in food consumption and activity to complete inappetence and reluctance to move at all from the perch or floor of the poultry house.

Respiratory symptoms were the most constant and obvious feature of the disease and comprised moist râles, sneezing, coughing and breathing through the partly open beak. A few fowls showed marked dyspnoea which took the form of a gasping type of inspiration with the head and neck outstretched, and some of these birds emitted a high-pitched noise coincident with these forced inspirations. This dyspnoeic type of breathing was, however, absent from some flocks and was never seen in more than 10 per cent. of the fowls in any one flock. It sometimes occurred within a few days of the initial symptoms but in other cases developed only after other signs of the disease had subsided. In a few fowls it was still present as long as 6 months after the outbreak of infectious laryngotracheitis had ended. The coughing up of blood was exceptional and was seen on only 2 farms, and then in only a few of the affected birds.

Conjunctivitis affecting one or both eyes was a common sign of infectious laryngotracheitis and was often accompanied by a watery or frothy exudate which was seen particularly at the anterior canthus. In some cases this exudate became mucopurulent and caused the eyelids to become adherent. Swelling of the eyelids, particularly the lower, was sometimes observed, and in a small proportion of cases there was marked oedema and inflammation of the lid. Ptosis of the lower lid was not uncommon, the affected eye appearing larger than normal. Peri-orbital oedema and swelling of one or both infra-orbital sinuses was occasionally seen. Lachrymation was rare. In a few birds the conjunctivitis was followed by keratitis, a caseous exudate adhering to the cornea with sometimes permanent opacity and loss of vision.

Nasal exudate, at first watery and later mucopurulent, was less common than respiratory or ocular symptoms but was seen in more than half of the birds. While some birds showed all these clinical signs concurrently, others showed only some of them.

Yellow caseous diphtheritic lesions were seen adherent to the roof and floor of the mouth and pharnyx at the base of the tongue and the rim of the larynx. A foul odour often accompanied these lesions.

A striking observation was that an occasional bird which had died of the disease was found dead on its back with its legs in the air.

The appearance of the disease in a flock was usually fairly sudden. At first one or two fowls showed ocular or respiratory symptoms and then a rapidly increasing number became affected, until all were showing symptoms after 7 to 10 days. A few birds were found dead without having shown any previous respiratory signs. Symptoms in individual birds usually lasted from 4 to 9 days, but sometimes up to 3 weeks elapsed before recovery or death occurred. By this time most of the birds which recovered were normal and bright in appearance, although some continued to sneeze and cough or show conjunctivitis for a variable period.

Egg production was reduced, even when mortality was low. When the disease occurred in birds during severe weather or late in the laying period they frequently went into a partial or complete moult, whereas birds affected earlier in lay and in milder weather returned to normal egg production within 3 weeks of the initial symptoms.

There was sometimes loss of weight among the affected stock. Morbidity was high and, as far as could be ascertained under field conditions, all the susceptible stock became affected. Mortality, on the other hand, not only varied from one outbreak to another but also from pen to pen on the same farm, and did not appear to be associated with the age of the affected fowl or the stage of the outbreak on the premises. It averaged 10 to 15 per cent. of affected stock with variations in flocks from 0 to 50 per cent. The highest death rate occurred in a flock of young cockerels which were also showing symptoms and lesions of vitamin A deficiency, a fact which was confirmed biochemically. A mild form of infectious laryngotracheitis, at first thought to be coryza, was seen in 2 flocks of 250 and 50 young birds respectively, reared on premises free of infectious laryngotracheitis but brought on to a farm where the disease was occurring among pullets. No deaths occurred although all birds showed mild respiratory symptoms. Because the disease was so mild and thought to be coryza no attempt had been made to isolate the virus of infectious laryngotracheitis from the birds, but 4 weeks after the commencement of the outbreak antibodies to the virus could be demonstrated.

Because of the similarity in the respiratory symptoms between infectious laryngotracheitis and Newcastle disease it is of interest here to record the early clinical features associated with an outbreak of Newcastle disease in a flock of pullets on a large poultry farm where infectious laryngotracheitis was being studied. After the initial observations and suspicion of Newcastle disease in this flock no further visits were made to the farm, and the writer is indebted to the poultry farmer for the further details. The disease was observed in a pen of 70 Brown Leghorn pullets which had not shown symptoms of infectious laryngotracheitis. The onset was sudden, sneezing and coughing being the most obvious signs, although lachrymation and nasal discharge were also seen. All the birds appeared to be affected at the same time. The symptoms were at first mild, but within 48 hours became severe and were accompanied by marked depression and complete inappetence. Within 1 to 2 days the droppings became increasingly fluid and of an offensive odour. Egg production dropped by 50 per cent. within 24 hours, and within 5 days the pullets ceased laying. The eggs laid during this time were either soft-shelled or of very poor shell texture and were laid indiscriminately about the poultry house. There were no deaths and after 8 to

9 days the birds were much improved and eating normally, but were not laying. By this time Newcastle disease had been confirmed by blood examination and the stock had to be slaughtered.

Epidemiology

Species susceptible: The fowl (*Gallus domesticus*) was the only species found to be affected with infectious laryngotracheitis. Turkeys and turkey poults were present on 3 of the farms during an outbreak and for at least 4 months afterwards but, despite the spread of the disease among the fowls and ample opportunity for indirect spread of infection, they remained healthy. Ducks and geese present on one farm during an outbreak of infectious laryngotracheitis in the fowls remained healthy. A large number of pigeons and budgerigars kept at one farm showed no evidence of disease despite an outbreak of infectious laryngotracheitis in the fowls which lasted 2 months.

Season: The disease was encountered at all times of the year; 3 outbreaks began in the spring (March to May), 5 in the summer (June to August), 2 in the autumn (September to November), and 1 in the winter (December to February).

Source of infection: In 9 of the 11 outbreaks it was likely that survivors from a previous outbreak were the source of infection; in one it probably spread from infected stock on a contiguous farm, and in another its origin could not be determined.

In 6 of the 9 outbreaks believed to be caused by survivors these birds had been reared on the premises, while in the other 3 outbreaks they were purchased from other farms; in 2 cases the birds actually came from the same farm. In all 9 outbreaks the survivor fowls, which are presumed to have been the source of the infection, remained healthy but a random sample of their sera was found to contain neutralising antibodies to infectious laryngotracheitis virus.

Spread of infection: Some interesting observations were made on the appearance of infection after contact between susceptible and survivor stock on a farm, and on the spread of infection from flock to flock among susceptible stock. On one farm 50 6-month-old survivor pullets were purchased and placed at one end of a battery containing susceptible pullets. Within 15 days the susceptible stock were showing symptoms of infectious laryngotracheitis. On a second farm, 25 survivor hens purchased in the market were placed in a deep litter house with 150 susceptible hens; signs of infection occurred in the latter 11 days later. On a third farm, 100 point-of-lay pullets were purchased and placed in one half of a large battery house partitioned with wire netting, the other half housing 200 susceptible pullets. The susceptible pullets showed symptoms of infectious laryngotracheitis 7 weeks later. Finally, on a fourth farm, susceptible pullets were placed in pens surrounding a very large pen containing stock which had survived a previous outbreak. Only the wire-netting fence separated the susceptible and survivor stock but symptoms were not seen in any of the susceptible pullets until 10 weeks later when the fowls in one pen became clinically affected. On this same farm young susceptible cocks were introduced into pens with survivor hens. The cocks in 3 pens showed clinical symptoms 11 to 15 days later.

Although the spread of infection among susceptible fowls within pens was quite rapid, the spread from one susceptible pen to another on the same farm was often comparatively slow, and in some cases several months elapsed before all the susceptible pens had been affected. For instance, on one farm in which 1,000 susceptible pullets and cockerels were housed in 15 semi-intensive pens and one deep litter house, 9 weeks elapsed between the appearance of the disease in the first pen and its appearance in the last, despite the fact that the pens were contiguous. In some outbreaks a lapse of a week or two occurred between the disappearance of the clinically recognisable disease from one flock and its reappearance in another. Spread from one susceptible flock to another on the same premises seemed to follow no definite pattern and disease in one flock was sometimes followed next by disease in a flock far removed from it.

Management and Housing: The disease was seen under deep litter, battery, semi-intensive and free range conditions. Except for 2 farms, the management was of a high standard, with no mixing of age groups or overcrowding of stock.

Post-mortem appearances

Observations were made on the carcases of over 70 fowls which had died of infectious laryngotracheitis within 7 to 10 days of showing symptoms. The birds varied from 7 weeks to 2 years of age and were autopsied within 24 to 48 hours of death.

Infectious laryngotracheitis was confirmed by the isolation of the virus. All organs and tissues were examined macroscopically. Despite the frequency of panophthalmia during life *post-mortem* examination of the eyes was of little value because of rapid decomposition.

In most cases the carcases were in good condition; some were fat, and many of the adult hens and pullets had recently been in full lay. There was no marked or widespread congestion of the carcase.

The most constant site of lesions was the respiratory tract. In almost every case there was congestion or inflammation of the trachea. Swelling and oedema of the mucous membrane were frequently seen, but the colour varied, being pale pink in some and deep red in others. In a few cases there were petechial haemorrhages. Diphtheritic changes were common and in many of these cases portions of necrotic tissue had sloughed into the lumen of the trachea while in others the necrotic mucous membrane of the whole of the trachea and primary bronchi was found as a cast in the lumen. The trachea constantly contained an exudate which was sometimes watery but more often mucoid. Caseous material was often seen lying free in the exudate and often causing complete obstruction of the lumen. This was particularly noticeable in the first inch of the trachea posterior to the larynx and also in the region of the syrinx. Caseous exudate adherent to the mucous membrane was a common finding in the larynx, where it often caused occlusion

of the lumen. The mucous exudate was sometimes slightly bloodstained but tracheal blood clots were rare.

The primary bronchi frequently showed similar inflammatory changes, and occasionally caseous exudate in the form of a plug occluded the lumen of one or both bronchi. Sometimes there were no gross lesions in these structures.

The lungs were less frequently affected than the upper respiratory tract but were sometimes congested, and mucus with or without particles of caseous exudate was seen in the primary and sometimes in the secondary bronchi.

In a few cases a small amount of semi-solid caseous exudate was found in the air sacs, particularly the thoracic, although the cervical, clavicular and abdominal were also sometimes affected.

The frequency of occurrence of macroscopic lesions in the respiratory tract may be seen from the following table prepared from the results of autopsy on 70 birds which had died of infectious laryngotracheitis. Lesions were found in all of them.

Total number of carcases examined	Site and frequency of lesions			
	Trachea	Primary bronchi (extra-pulmonary)	Lungs	Air sacs
70	69 (20)*	50 (1)	29 (0)	9 (0)

* The figures in brackets indicate that lesions were found in that site only.

It will be seen that lesions diminish in frequency from the trachea which was affected in 69 out of 70 cases to the air sacs which showed lesions in only 9. The trachea alone was affected in 20 cases and the primary bronchi alone in one.

Caseous material was occasionally seen in the mouth as plaque-like bodies varying in diameter from that of a pin head to 3 to 5 mm., loosely attached to the mucous membrane of the fauces, larynx, roof of the mouth or under the tongue, and often extending into the first one to two inches of the oesophagus. Sometimes, however, it appeared to be diphtheritic in nature and was more firmly adherent to the underlying tissue.

Diagnosis

In the outbreaks studied the clinical signs, spread and course of disease, and the *post-mortem* appearances were helpful, but not sufficiently specific to afford a definite diagnosis of infectious laryngotracheitis, which depended upon the isolation of the virus.

Isolation of the virus: This will be reported in detail elsewhere (Jordan, 1958) but the following is a brief account of the work.

Material from both live birds and carcases was examined. In live birds swabs were taken from the trachea, conjunctiva and nasal exudate. In carcases the trachea and conjunctiva were swabbed, and a small portion of the lung containing the primary bronchus was removed. When air sac lesions were present they were also examined for virus. Swabs from live birds were examined within 48 hours, and material from carcases was examined within 48 hours of death.

Each swab was broken into a separate bijou bottle containing 1 ml. of nutrient broth incorporating 1,000 units each of pencillin and streptomycin per ml. The bottle was vigorously shaken and allowed to stand for half an hour at room temperature. If a fairly clear supernatant fluid was not readily obtained in this way the material was centrifuged at 3,000 r.p.m. for 10 minutes.

Portions of lung or air sac wall were ground in a Griffith tube with sterile powdered glass and 1 to 2 ml. of nutrient broth which incorporated penicillin and streptomycin as above. The fluid was then pipetted into a bijou bottle and centrifuged as for swabs.

The supernatant fluid from material from each site was inoculated into a separate series of four 10- to 12-day-old embryonated eggs, an inoculum of 0.1 ml. being pipetted on to the dropped chorioallantoic membrane of each egg. The eggs were then incubated at 37° C. for 3 days when the dropped chorioallantoic membrane was removed and examined for pocks.

As pocks are produced on the chorioallantoic membrane by viruses of both infectious laryngotracheitis and fowl pox it was necessary to differentiate them by swabbing the affected chorioallantoic membrane, after trituration, on the scarified comb of a susceptible fowl. By this method infectious laryngotracheitis virus was detected in live birds and in carcases from all 11 outbreaks. No evidence of fowl pox was found in any of them.

In order to determine from which site infectious laryngotracheitis virus could best be recovered, 41 live affected fowl and 25 carcases from field cases were examined. The virus was isolated from 17 of the live birds, each of which had been swabbed within 6 days of first showing symptoms. The virus was isolated 12 times from the trachea, 11 times from the conjunctiva, 5 times from the trachea alone and 3 times from the conjunctiva alone. Nasal exudate was present in 10 birds and the virus was isolated from it on 3 occasions but never from this site alone.

Virus was isolated from 20 of the 25 carcases. Eighteen of the 20 birds had died within 6 days of initial symptoms, and the other 2 after 20 and 48 days. The lungs, trachea and conjunctiva were examined from each carcase and the virus was isolated from these sites 16, 15 and 13 times respectively. On one occasion the lungs were the only site from which the virus was found. Lesions were present in the air sacs in 5 carcases and from 4 of them the virus was isolated. In 2 of these it was the only site from which the virus was obtained.

Discussion

The clinical appearance of the disease in these outbreaks was much less severe than that recorded by Dobson (1935a, b), who described the coughing up of blood and bloodstained mucus as a frequent symptom. Seddon (1952) in Australia has classified infectious laryngotracheitis on clinical and *post-*

mortem appearances into 4 main types which tend to merge into one another. They are: (1) peracute (high morbidity and mortality of 50 to 60 per cent. or more, sudden illness, coughing up of blood and bloodstained mucus); (2) acute (high morbidity but low mortality, 10 to 15 per cent. haemorrhagic tracheitis rare); (3) chronic or mild (this type comprises survivors of the previous types—morbidity is low, 2 to 5 per cent.—and the chief symptoms are unthriftiness, coughing and gasping respiration and a foetid nasal discharge) and (4) an asymptomatic type. The outbreaks described by us fall into the acute type of Seddon in that this form was seen in all of them, although a mild form of the disease with coryza-like symptoms and low mortality, not easily recognised as a type of infectious laryngotracheitis or readily classified into Seddon's 4 types, was seen in 2 pens on one farm.

Whilst the 11 outbreaks seen by us may be typical of the type of infectious laryngotracheitis prevalent in Cheshire and Lancashire it must be pointed out that they were largely selected in that the poultry farmers sought advice because birds were dying with a respiratory disease, and 8 of the outbreaks were suspected of being Newcastle disease. If a more severe form of infectious laryngotracheitis occurred in the area it would similarly have been brought to our attention but it may well be that milder outbreaks simulating coryza, with little or no mortality and unaccompanied by the more severe form, would not have been reported. Whether a mild form of infectious laryngotracheitis without any deaths occurs merits further investigation.

The *post-mortem* appearance of fowls which died of infectious laryngotracheitis differed from that described by Dobson (1935a, b) in that in our experience generalised congestion of the carcase and marked haemorrhagic tracheitis with blood clot in the lumen of the trachea were rare. Unlike Dobson, and also Seddon (1952), we frequently found diphtheritic, caseous and mucous exudate extending from the trachea into the primary bronchi. In nearly half our cases it extended into the smaller bronchi and in a few cases into the air sacs. Extension of exudate into the primary bronchi was observed by Beach (1926) and Seifried (1931), the latter observing pulmonary lesions in a few birds. Except in the peracute form of the disease, however, it is difficult to ascertain to what extent the lesions are due to the virus of infectious laryngotracheitis alone and to what extent other viruses, bacteria and deficiency conditions may contribute to their production. In some cases lesions of infectious laryngotracheitis may accompany those of another respiratory disease.

The usual cause of death in infectious laryngotracheitis has been considered to be obstruction of the respiratory tract (Beach, 1926 and Beaudette, 1937) and our findings confirm this, but there were a few cases where it was difficult to ascribe the cause of death to asphyxia.

As Delaplane (1945) and Seddon (1952) have pointed out, only the peracute types of the disease can be diagnosed on symptoms and lesions alone. The type of disease we have described may be confused clinically and at autopsy with several other diseases. For example the symptoms may simulate the respiratory form of Newcastle disease although the spread of infectious laryngotracheitis on a farm may not be as rapid. It should be emphasised that when dealing with respiratory conditions the existence of Newcastle disease should first be eliminated. In some fowls infectious laryngotracheitis might be mistaken for chronic respiratory disease, coryza, infectious bronchitis, vitamin A deficiency and, in fact, any of the respiratory diseases of poultry. There is the further complication of more than one of these diseases occurring simultaneously. In a flock outbreak, fowl pox can be differentiated by the presence of comb and skin lesions, but the two diseases may co-exist.

The source of infection in 10 of the 11 outbreaks was infected stock which were apparently healthy. This is in agreement with Seddon (1952) who indicated that contact of susceptible with clinically affected or carrier birds was a common means of spread.

That there is considerable excretion of the virus during the first week of symptoms is shown by the rapid spread of disease within the flock, and the ease with which the virus can be isolated during this period. On the other hand, the infrequency of isolation of the virus after this time, and the length of contact between susceptible and survivor stock before disease occurs (on one farm up to 10 weeks) suggest that only a small proportion of survivors excrete the virus, and then perhaps only intermittently, as was observed by Scott and Brandly (1934).

The published evidence for the persistence of infection in survivors suggests that few become carriers but that some of them may excrete the virus long after recovery. Komarov and Beaudette (1932) were able to demonstrate virus in tracheal swabs of 6 of 8 birds 72 days after the natural disease had subsided, and in 3 of 25 similar birds 16 months after recovery. Gibbs, quoted by Beaudette (1949), found 14 carriers among 504 birds examined and later found 22 birds excreting the virus out of about 1,000 survivors. One bird was still a carrier 741 days after infection. Seddon (1952) states that most affected birds recover completely but that some become carriers and may remain infective for more than 2 years.

The irregular pattern of spread of infection from pen to pen on a farm is similar to that noted by Gorrie (1945).

The control of infectious laryngotracheitis may be considered along 4 lines—(1) prevention of the introduction of the infection; (2) eradication; (3) treatment of sick birds and (4) vaccination.

(1). Since Komarov and Beaudette (1931) and Brandly (1934) have shown that the virus is unlikely to be spread in or on the egg, and since Seddon (1952) reports that the danger of spread by day-old chicks is negligible, the introduction of infection on to a farm could be avoided by purchasing only day-old stock or hatching eggs. An alternative method would be to purchase older birds from sources known to be free from the disease.

(2). All survivor stock on a farm might have been

infected even if symptoms were mild or absent and, although only a proportion of such birds will be carriers, it is not practicable to distinguish them from recovered birds which have freed themselves from virus. All survivor stock must therefore be considered potential carriers. In view of this, and since the virus does not appear to survive long outside the host (Seddon, 1952), and since it is readily destroyed by the common disinfectants (Schlam & Beach, 1935) a logical means of control is to dispose of all survivors and cleanse and disinfect the premises before restocking. As long ago as 1933 such a plan was proposed by Gibbs (1933). This plan comprises the removal of all survivor stock, cleansing and disinfection of houses and equipment, and resting the runs, houses and equipment for 2 months before restocking. An attempt at eradication along these lines is being undertaken under our supervision by a farmer who has suffered outbreaks of infectious laryngotracheitis in his stock during the last 2 years. He is a pedigree breeder and is anxious to keep his strain of fowl. All stock on his premises is to be sold at the end of the hatching season and the buildings and equipment disinfected. The replacement stock will be birds which had been removed at day old to uninfected premises a mile away, reared in isolation and returned not less than one month from the time of disinfection.

(3). There appear to be no reliable records in the literature of the treatment of birds affected with infectious laryngotracheitis and no attempt has been made by us to assess the value of chemotherapy under controlled conditions. In 6 of our 11 outbreaks, however, chlortetracycline,* oxytetracycline† or sulphadimidine was used. On 4 farms chlortetracycline was fed at the rate of 200 g. per ton of food for 14 days as soon as disease was seen in a pen and at half this strength to all susceptible birds. There was no obvious alteration in mortality, course of the disease, or spread of infection among the treated stock when compared with untreated stock on other farms. Oxytetracycline was similarly fed on 2 farms at a rate of 200 g. per ton of food without obvious beneficial effect. On 2 of the 4 farms where chlortetracycline was fed sulphadimidine was also used. A 0.2 per cent. solution was given in place of the drinking water for 6 days as soon as symptoms were seen, using the routine advocated by Davies and Kendall (1954) for the treatment of coccidiosis. It did not appear to influence the disease in any way.

(4). In countries where the disease is endemic widespread vaccination is practised to reduce losses and also to improve production in flocks affected with the chronic type of infectious laryngotracheitis. A disadvantage of vaccination is the possible risk of producing carriers. While Nicol (1945) and Beaudette (1949) state that vaccination does not give rise to carriers, Gorrie (1945) and Pulsford, Chamberlain and Topham (1956) believe that vaccinated birds may be a source of infection. Because of this risk vaccination in this country cannot be recommended unless the infection proves to be widespread and to cause serious losses.

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* Aureomycin. † Terramycin.

PREVALENCE OF MILK FEVER

A farming journal writes:—

From the north-east we learn that milk fever among cows and heifers is still seriously prevalent. In some areas of Aberdeenshire and Banffshire veterinary surgeons have been "plagued" with the numbers of cases occurring in the last 2 or 3 weeks. Within a 12-mile radius as many as from 20 to 30 cases have had to be dealt with, and calcium injection has not always been effective.

In the case of heifers going down, the theory is that poor wintering and the rough spring when they carried their calves had reduced the calcium content of their bodies.

Once more it is observed that the incidence of the trouble was most pronounced on highly-farmed land. In one such case ten animals required treatment on the same day. Has pasture husbandry moved too far ahead of the capacity of the animal body?

Progressive Muscular Dystrophy in the Dog

BY

J. C. WHITNEY

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SUMMARY.—The clinical history, post-mortem findings, and histopathology of the muscle lesions in three cases of progressive muscular dystrophy in the dog are recorded. The aetiology and relationship of this condition to similar conditions in man and animals are discussed. The condition appears to be similar to restricted muscular dystrophy of the limb-girdle type in man and bears little relationship to the muscular dystrophies of the ox and sheep.

Introduction

THE study of this condition comprises the examination of 3 cases which were presented for *post-mortem* examination in this Department during the period January to June, 1957. It should be stressed at the outset that in only one case was there a clear history of lameness (Case III); in one other case there was a vague reference to slight weakness of the hind limbs (Case II) and there was no history of lameness in the third (Case I).

There is only one previous report of muscular dystrophy in the dog by Innes (1951). "The subject was a 9-year-old retriever dog which showed slow, progressive stiffness of the hind limbs over a period of 6 months. In the last 2 months, the condition declined rapidly. The dog was restless and appeared to be in pain and was very overweight with a diffuse bilateral cataract and atrophic, flabby testicles. The hocks in both hind limbs were lowered almost to the ground and suggested section of the Achilles tendon. At *post mortem*, the gastrocnemius muscles in both limbs showed a distinct pallid colour. All other muscles of the hind limbs were normal. Upon histological examination, there was marked atrophy of the whole fasciculi to the point of complete disappearance of many fibres and replacement with adipose tissue. Very few lymphocytes were scattered between atrophic fibres. There was pronounced proliferation of sarcolemmal nuclei. Blood vessels and peripheral nerves were normal."

Clinical and *Post-mortem* Findings

Case I

The first case (P.M. 130/57) was in a 12-year-old Sable Pekingese dog which was presented in the Clinical Department as a case of congestive heart failure. At *post-mortem* examination the dog showed a chronic passive congestion of the liver, lungs, and spleen. The medial tricuspid and anterior and posterior mitral valves showed lesions of a chronic valvular endocarditis. The semimembranosus and semitendinosus muscles in both hind limbs were pale and slightly atrophic when compared with the associated muscles in those limbs. The histological appearance of these muscles will be discussed below.

Case II

In April, 1957, a 10-year-old Labrador bitch (P.M. 567/57) was presented for *post-mortem* examination. The previous history in this case was that in September, 1956, there had been a slight vaginal discharge and evidence of cardiac incompetence. In March, 1957, she showed slight weakness of the hind limbs and excessive thirst. In April, 1957, she was presented showing signs of pyometra with an associated cardiac incompetence. The bitch was considered a bad surgical risk and it was decided to destroy her. At *post-mortem* examination it was noticed that the semitendinosus and semimembranosus muscles of both hind limbs were paler than normal, showing faint grey streaking which contrasted strongly with the rich, red colour of the associated muscles of the hind limbs. The muscles were not atrophic and felt full and firm in consistency. The histological appearance of these muscles will be discussed below. Additional *post-mortem* findings in this bitch were of cystic ovaries associated with hormonal pyometra, cystic nodular hyperplasia of the adrenal cortex, a right inguinal hernia containing a loop of the right uterine cornu, and chronic valvular endocarditis of the mitral and medial tricuspid valves. The bitch was in excessively fat condition.

Case III

Case III was a 10-year-old Alsatian bitch (P.M. 1193/57) with a history of weakness of the hind limbs for 2 weeks. The condition became rapidly worse and when examined in the Clinical Department in June, 1957, the dog was markedly lame, being unable to get the right hind limb forward beyond the verticle, the toes being in flexion. Thus, the leg invariably collapsed when weight was applied, although at rest the dog was able to stand. The muscles of the hind limb, the vastus medialis, vastus lateralis, and rectus femoris were markedly wasted and flabby. The left hind limb was weakened but there was more muscular development. X-ray examination of the lateral lumbar spine and the dorso-ventral lumbar spine and pelvis showed no abnormality. A diagnosis of femoral paralysis of the right hind limb was made and the dog was destroyed. *Post-mortem* examination showed that the extensors of the hind limb, the vastus lateralis, vastus medialis, and rectus femoris were pale and atrophic. The flexor muscles were of normal size and rich red in colour. The extensors of the left hind limb, although pale in colour, showed less pronounced lesions than those on the right side. No significant macroscopic lesions were observed in either the femoral nerves or in the lumbar cord.

Additional *post-mortem* findings in this case were as follows:—

A well differentiated adenoma of the left thyroid gland which measured 4.1 by 2.1 cm.; a small heart base tumour, 0.8 cm. in diameter; and a cystic hyperplasia of the endometrium of the uterus, associated with cystic follicles in both ovaries.

Histopathology

Staining Methods

Blocks of muscle were fixed in 10 per cent. formol saline. Some were processed by frozen section technique and stained Sudan IV. The remainder were processed in paraffin wax and stained by Harris's haematoxylin and eosin. The sections of muscle were also stained by Heidenhein's haematoxylin and eosin and by Masson's trichrome stain; paraffin sections of the femoral nerve and spinal cord from Case III were stained by Holmes's silver method for neurofibrils and by Loyez haematoxylin for myelin sheath.

The histopathology in all 3 cases was identical to that in Innes's (1957) case, and the principal changes are summarised below:—

The muscle fibres were atrophic and the bulk of the muscle was restored by adult fat cells which had appeared between them (Plate III). The muscle fibres varied in size and a minority were hyaline and homogeneous with blurring of their striations (Plate II). There was no increase in the connective tissue (Plate I). The sarcolemmal nuclei were proliferated especially in Case III. These nuclei varied in size and shape and some were centrally placed within the muscle fibres (Plate IV). A sparse lymphocytic infiltration was observed between the atrophic muscle fibres and around a few small venules in Case III (Plate IV). No changes were observed in the nerve fibres in the muscle sections or in sections of the spinal cord and femoral nerve in Case III.

In Case I blocks were taken from the affected muscles only. The lesions in Case II were confined to the semimembranosus and semitendinosus muscles in both hind limbs. Sections from related muscles in these limbs showed no abnormality. No blocks of muscle were taken from other parts of the body. In Case III the lesions were confined to the vastus group and the rectus femoris in both hind limbs. Sections from the head, shoulder, lumbar, and flank muscles showed no abnormality. The sections from the other muscles of the hind limbs showed no abnormality.

The muscle lesions were bilateral and similar in all three cases.

Discussion

A muscular dystrophy is, by definition, a primary degenerative disease of skeletal muscle. The innervation of affected muscles in contrast to the neural and spinal atrophies is sound. There is a symmetrical distribution of the muscle lesions and a retention of faradic excitability in proportion to the remaining power of contraction. Cutaneous reflexes are retained and there is a marked heredofamilial incidence (Adams *et al.*, 1954).

This definition of muscular dystrophy is used in human medicine and whether we can regard muscular dystrophy in animals as analogous to that in man is open to question, but these cases would seem to fit into this category in its broadest concept. It is considered that the histological appearance of muscular dystrophy is not specific but is the same in all clinical forms of the disease and, thus, should be regarded as clinico-pathological entities.

Innes regarded his case as identical with human cases of true progressive muscular dystrophy of the pseudohypertrophic type. This description presumably refers to the severe generalised familial muscular dystrophy of man in its most common form as Duchenne's pseudohypertrophic muscular dystrophy (Adams *et al.*, 1954). In man, this condition has a strong familial liability, occurring predominantly in the male (2 of the 3 cases recorded here were female). The condition is said to be due to a sex-linked recessive gene carried by the female but manifested in the male. Symptoms are usually manifest by the sixth year and the condition is characterised by enlargement of the muscles, particularly those of the lower limb. Later, the affected muscles become atrophic (Walton, 1957).

There was no history or evidence of severe lameness or muscle atrophy in Cases I and II, and thus they may be regarded as pre-clinical or early clinical cases. Consequently, one might expect some enlargement of the muscles if these cases were comparable to human dystrophy of the Duchenne type, but none was observed.

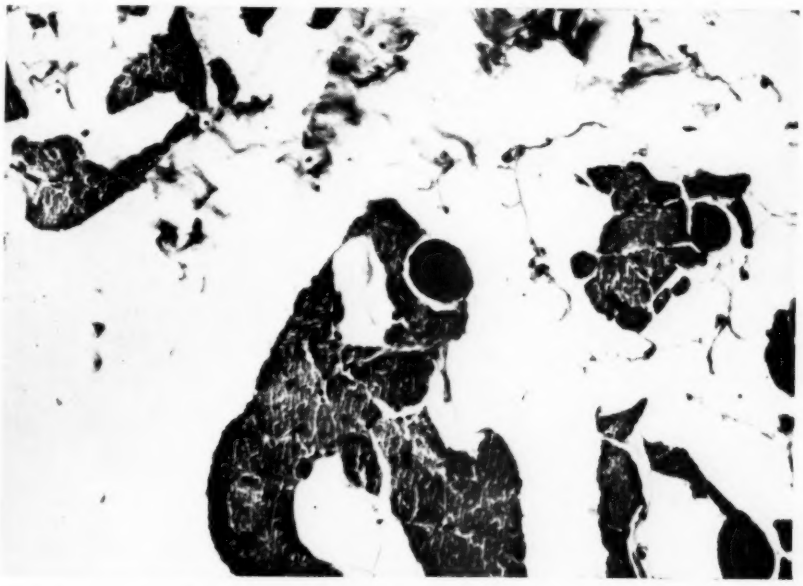
The ages of these 3 dogs ranged between 10 and 12 years. It has not proved possible to trace the origin of these dogs, but it is suggested that it is unlikely that this condition was manifest in early life. Thus, bearing in mind the age of the subjects, the absence of pseudohypertrophy in any of the affected muscles and the absence of the sex predisposition, it is felt that the term "pseudohypertrophic muscular dystrophy" is misleading, as it tends to be associated with the human condition. It is suggested that the term "progressive muscular dystrophy" would be more appropriate.

Another type of muscular dystrophy which has been reported in man, restricted muscular dystrophy of the limb-girdle type, occurs in adults or adolescents, affecting either sex equally. The muscles of the upper or lower limbs may be affected. The condition may become arrested at any point and rarely causes severe disability. Pseudohypertrophy is not a feature of this condition, although compensatory hypertrophy may occur in related muscles. It is considered by some workers that this condition is due to an autosomal recessive gene. These 3 cases in the dog are similar to muscular dystrophy of the limb-girdle type.

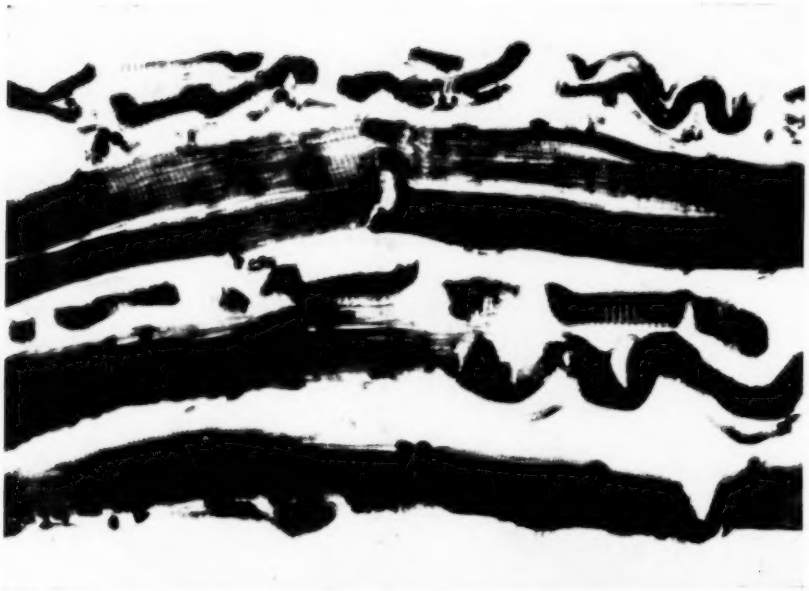
With reference to muscular dystrophy in other animals, by far the most common and economically important type is the degeneration which occurs in cattle and sheep associated with a deficiency of vitamin E. There was no opportunity to assess the effect of vitamin E therapy in any of these dogs but it is unlikely that it would have proved beneficial.

J. C. WHITNEY.—PROGRESSIVE MUSCULAR DYSTROPHY IN THE DOG

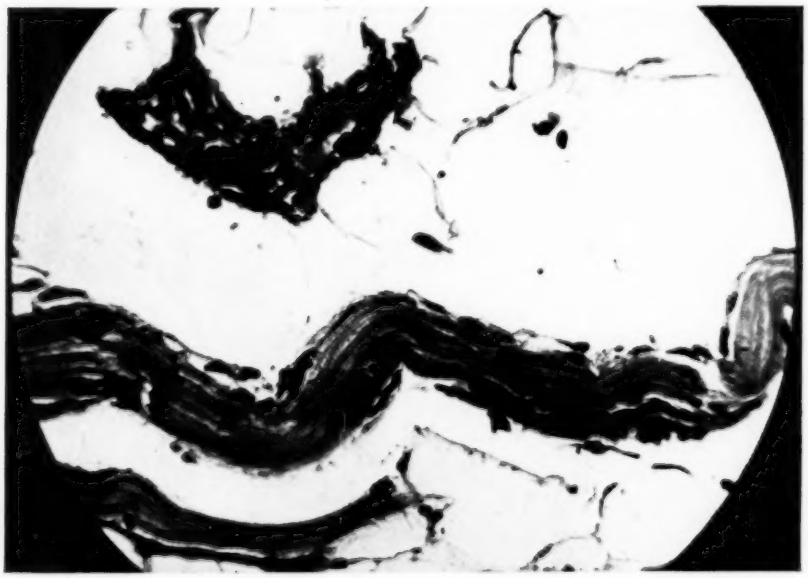
Longitudinal section—Semitendinosus muscle of a 10-year-old Labrador bitch (Case II). Showing atrophy of the muscle fibres and replacement by fat cells. There is no increase in fibrous tissue in the perimysium. (H. & E. × 80.)



Transverse section—Semitendinosus muscle of a hind leg of a 12-year-old Sable Pekingese dog (Case I). The section shows a great variation in the size and shape of muscle fibres. Some fibres are split into their component myofibrils. Two homogeneous hyaline muscle fibres are observed in this field. (H. & E. × 260.)



Longitudinal section—Vastus group from the right hind leg of a 10-year-old Alsatian bitch (Case III). This section shows persistence of striation in muscle fibres, showing advanced lesions of progressive muscular dystrophy. (Heidenhain's haematoxylin × 750.)



Longitudinal section—Vastus group of the right hind leg of a 10-year-old Alsatian bitch (Case III). The section shows a perivascular lymphocytic infiltration around the venule and the proliferation of sarcolemmal nuclei. (H. & E. × 700.)

Other types of muscular dystrophy have been described in cattle and sheep by various workers, but these do not appear to bear any relationship to the cases which occur in man or in the dog (Muth, 1955; Graham Marr *et al.*, 1956; Dodds, 1954; Parry, 1957; Blaxter, 1957).

Some features of the clinical history in Innes's case of testicular atrophy, bilateral cataract, and muscular atrophy suggest that this might be similar to a late case of Dystrophia Myotonica, a rare human disease of dominant inheritance which occurs in the adult. The presence of dystrophic changes in the gastrocnemius muscles in this case precludes a diagnosis of congenital myotonia (Thomsen's disease). Congenital myotonia has been observed in certain strains of goats (White & Plaskett, 1904; Brown & Harvey, 1939).

In Case I there was no history of lameness and and any locomotor disturbance that may have arisen probably would have been ascribed to the heart condition that was present, had the dog survived. Histologically, the lesions were not advanced. Case II showed some slight weakness of the hind quarters which tended to sway during locomotion in the last few weeks of life. The involvement of the semimembranosus and semitendinosus muscles can be correlated with this history. Any loss of function of these muscles would reduce forward propulsion and interfere with adduction of the limb and extension of the hip and hock. The reduced ability to adduct the limb would result in the hind feet being held wider apart than normal, and give the dog a waddling, swaying gait, and the range of forward movement at each stride would be reduced. These symptoms were ascribed to the heart condition that was present and to the developing pyometra. The bitch was in excessively fat condition.

In Case III a diagnosis of femoral paralysis was made. The femoral or anterior crural nerve supplies the rectus femoris, the vastus group and the sartorius muscle *via* the saphenous nerve. Consideration of the distribution of the muscle lesions and the clinical history in this case showed that, in the absence of a muscle biopsy, it would have been very difficult to differentiate this case from a case of femoral paralysis.

The muscle condition in these three cases was diagnosed at *post-mortem* examination and thus no treatment was undertaken.

It is surprising, perhaps, that so few cases of progressive muscular dystrophy in the dog have been recorded in the literature when three of the four recorded cases came to *post mortem* in this Department during the 6 months, January to June, 1957. It would suggest that this condition is far more common than it appears and it is probable that many cases are diagnosed as spinal or neural atrophy. These conditions could be differentiated by muscle biopsy.

What is the significance of this condition in the dog? It is considered that the condition is important from the point of view of differential diagnosis. There is little hope of an effective cure for progressive muscular dystrophy but it may be possible to relieve the symptoms of muscular atrophy of nervous origin.

A second significant point lies in the field of comparative medicine. As there is no treatment for human myopathies, a study of the condition in the dog may throw some light on the aetiology and pathogenesis of the human disease.

Acknowledgments.—My thanks are due to Mr. I. S. Beattie for his guidance in the preparation of this paper and his approval of the final draft; to Mr. K. W. Head and Mr. R. C. James for the photomicrographs; and to Mr. J. Heeps for the preparation of the sections.

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Experiments in Cattle with *Trichomonas suis*

BY

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HAMMOND, Fitzgerald *et al.* in 1955 reported that infection with trichomonads originating from swine may be associated with infertility and abortion in cows, subsequently Hammond *et al.* (1957) reported infection of the bull by *Trichomonas suis*, also that this infection was transmitted by coitus to females and back again to males. *Trichomonas suis* was the name given to the trichomonad isolated from the stomach of the pig. The early cultures isolated by Hammond had been isolated from faeces and from the caecum. Whether there was any difference antigenically or physically between these strains at that time is not stated. A very great difficulty when isolating any culture of trichomonads is the problem of separating the trichomonad from its bacterial contaminants. At the same time one is confronted with the problem of educating the organism to grow in artificial media free from its bacteria and normal food supply. A useful method was fortunately found to overcome the first part of the problem and that was by adding either pigs' faeces containing trichomonads or a contaminated culture to semen which was streptomycin treated (.01 mega unit to 1 ml.). This semen was then used to inseminate a virgin heifer. Although the trichomonad organisms appeared at the 8th day in vaginal mucus, cultures were not made until the 24th day so as to allow sufficient time for the uterus to eliminate most of the bacterial contaminants.

(*Continued overleaf*)

The main purpose of these experiments was to examine further Dr. Hammond's work, to obtain the clinical picture of *Tr. suis* in cattle and to explore the possibility of making an efficient diagnosis by the use of the vaginal mucus test.

The cultures used were two strains of *Tr. suis* kindly supplied by Hammond, and a Stormont strain isolated from pigs at this laboratory. The method of infection is important. In our experience trichomonads added to semen and placed intrauterine at oestrus was the most effective method. However, one animal, H.6, was successfully infected intravaginally when not in oestrus. The number of trichomonads to be added to the inoculum is not of importance as in our experience heifers can be infected equally well with small as with large numbers. Normally 100,000 parasites in 0·1 ml. saline are added to semen immediately prior to the insemination. The method of cultivating the parasite from the uterus and the methods of vaginal mucus tests have been described (Pierce, 1947; Kerr & Robertson, 1941; and Kerr, 1955).

Heifer 1

0·1 ml. of a suspension of *Tr. suis* (Hammond) was added to the semen and the heifer inseminated. Two days later the vaginal mucus contained active trichomonads. The vaginal mucus showed active organisms for 42 days, numerous on some occasions and scanty in numbers in others which is a characteristic feature of trichomonad infections. Oestrus returned on the 42nd day and the heifer was inseminated with non-infected semen. From that day the vaginal mucus was positive until the 137th day when trichomonads ceased to appear but they were still shown by culture to be present in the uterus until the 191st day when the experiment was terminated. The most important clinical feature noted was that the uterus on the 118th day contained more than 600 ml. of fluid rich in cells and active trichomonads.

Heifer 2

Infected in a similar manner to H.1. On the 4th day the vaginal mucus showed active trichomonads and these were found intermittently for 82 days. Oestrus was noted at the 36th and 70th days.

In these two heifers a clinical trichomoniasis was induced which was as virulent and typical as that induced by any of our *Tr. foetus* strains. Microscopically it is impossible to differentiate *Tr. suis* from *Tr. foetus*. The mucus agglutination test results are discussed later.

Experiments with *Trichomonas suis* strain Stormont

Heifer 3

0.1 ml. of a suspension of pigs' faeces was added to semen containing streptomycin and placed into the uterus at oestrus. At 8 days numerous active trichomonads appeared in the vaginal mucus and persisted for 39 days. Subsequently their appearance was sporadic until the 71st day when oestrus occurred. Thereafter no trichomonads were found microscopically or culturally. At 142 days oestrus returned and the heifer was inseminated successfully.

Heifer 4

A passage experiment of *Tr. suis* strain Stormont from H.3 was carried out. Four days after insemination the vaginal mucus became positive microscopically and remained so until 46 days when oestrus ensued. The uterus at this stage contained about 600 ml. of fluid rich in trichomonads—a typical pyometra. The uterus was irrigated with 1/400 iodine. No evidence of organisms was found until the 63rd day when they reappeared in large numbers and were most active. Their appearance remained sporadic until the 162nd day when the experiment was terminated.

This was a very active clinical manifestation of the disease.

Clean heifers 5, 6 and 7 are passage experiments with *Tr. suis* (Stormont) again using infected semen intrauterine.

Heifer 5

Four days after insemination with infected semen the vaginal mucus was positive on microscopical examination. The discharge was rich in cells and trichomonads and remained positive for 34 days. Oestrus returned at 19 days and the heifer was inseminated and eventually produced a calf from this insemination although organisms were excreted for 15 days after the insemination.

Heifer 6

Same result as in H.4 and H.5. Produced an infection which remained for 106 days. Again sometimes samples were negative and at other times the organisms were plentiful. The heifer was infertile and did not prove in calf.

Heifer 7

Same result as in H.4, H.5 and H.6. After insemination the trichomonads appeared sporadically on microscopic examination for 71 days. The heifer was infertile for 4 months when conception took place at the 3rd insemination.

Heifer 8

Infected intravaginally with *Tr. suis* (Stormont). 10 ml. of suspension of organisms were placed in the vagina midway between the oestral periods. Trichomonads appeared at the 3rd day and were seen intermittently for 31 days. Oestrus returned at 10 days, 31 days, and 51 days and the heifer although inseminated at each oestral period did not prove in calf. Conception occurred at a subsequent insemination.*

Infection of pigs with *Tr. foetus* and *Tr. suis* (Stormont)

Sow 1 immediately after service was inoculated intravaginally with 10 ml. of a concentrated suspension of *Tr. foetus*. Trichomonads not seen in vaginal mucus at any stage during pregnancy.

Sow 2 inoculated intravaginally after service with *Tr. suis* (Stormont). Trichomonads were present at the 6th day and persisted for 8 days, thereafter they were not seen again. This sow had a normal litter.

*It is normally much more difficult to infect an animal intravaginally with *Tr. foetus* when it is not in oestrus than at the time of insemination. This heifer was successfully infected with *Tr. suis* when not in oestrus.

Two gilts were inoculated intravaginally with *Tr. suis*. One remained completely negative, the other showed trichomonads for 7 days which would suggest that some multiplication had taken place in the vagina. The vaginal mucus thereafter remained negative.

A poor response was obtained in the female pig with the pig strain of trichomonad. It may be that the genital system of the pig has a greater resistance than that possessed by the cow. In any case the author has never found in the field a trichomonad infection in the pig although it is a common inhabitant of the intestinal tract of this animal.

Diagnosis

Direct microscopic

The appearance of *Tr. suis* in the vaginal mucus has been discussed in the history of the heifers. They may be plentiful at some examinations and scanty in numbers at others but this is typical of trichomonad infection. A negative test is suspect in an infected herd. As stated before it is not possible to differentiate *Tr. suis* and *Tr, foetus* microscopically.

The cultural method is a useful adjunct particularly at the end of an infection when cultures may be positive for some time after the vaginal mucus is negative (Heifers 4 and 1).

Mucus agglutination test

This test which has given such good results in the field control of *Tr. foetus* was used to determine if *Tr. suis* was closely enough related to it to elicit a positive reaction. It is not proposed to enter here into the details of the numerous tests carried out with the different strains against the various mucus samples and uterine washes as a detailed study of the antigenic picture of this organism will be published by Dr. M. R. Robertson of the Lister Institute. In trichomoniasis in cattle two strains have been recognised, Belfast and Manley (Kerr & Robertson, 1945). Belfast is much more commonly found than Manley which, however, is equally virulent; the two strains are serologically distinct. *Tr. foetus* Belfast and *Tr. foetus* Manley share many antigens but they do not share the specifically important polysaccharide antigens to any effective degree and so are serologically distinct. Bovine vaginal mucus which contains antibodies is less critical in agglutination than the serum of the rabbit which has been sensitised intravenously to trichomonas. Therefore rabbit serum is more valuable for critical analysis than the mucus test. An example of the relationship of the strains when subjected to a vaginal mucus test is given below:—

H.4 vaginal mucus (4.2.57) was tested against *suis*, Belfast and Manley.

	1/10	1/20	1/40	1/80	1/160	1/320
Belfast suspension	++++	+++	++	++	+	±
Suis suspension	++++	+++	++	+	+	±
Manley suspension	+	±	-	-	-	-

In this test the Manley strain gives only a slight reaction, but the *suis* reaction is similar to that of Belfast. It is interesting to note that *Tr. foetus* Belfast and *Tr. foetus* Manley from the cow are serologically distinct, while *Tr. suis* from the pig and *Tr. foetus* Belfast are more closely related serologically as shown by the action of the vaginal antibody. Throughout the experiment all heifers reacted to the vaginal mucus and uterine wash agglutination tests and it can be accepted that trichomoniasis induced in cattle by *Tr. suis* can be adequately tested by using strain Belfast.

These experiments confirm Hammond's clinical experiments with *Tr. suis*, in that this organism infects cattle and produces a classical picture of trichomoniasis; moreover, the infection can be passaged from heifer to heifer without difficulty. These experiments also show that clinically *Tr. suis* cannot be differentiated from *Tr. foetus* nor can they be separated by the mucus agglutination test to which they both respond in a similar manner. The possibility of an outbreak of trichomoniasis in the bovine from porcine faeces cannot be ignored.

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O.E.E.C. EXPERTS DISCUSS THE APPLICATION OF ATOMIC SCIENCE IN AGRICULTURE

Leading specialists in the application of atomic science in agriculture and food, from European member countries and the United States, met at the O.E.E.C. headquarters in Paris on July 21st to 25th to discuss the latest advances resulting from research in their own countries and to plan further progress and dissemination of knowledge of this new approach to improved agricultural output.

This meeting is a further step in the programme of the O.E.E.C. European Productivity Agency, designed to ensure the efficient use of the application of this science and to stimulate co-operation in research programmes both within and among O.E.E.C. countries. Parties of European scientists have already made extended tours in the United States and the United Kingdom, and further visits of this kind are planned in the E.P.A. long-term programme. In addition, highly qualified American consultants have visited the Member countries of O.E.E.C. to investigate and advise on progress; they were among the principal speakers at the meeting.

The programme of the meeting was based on four main fields in which the use of atomic science in agriculture and food is being applied throughout Europe and in the United States.

In Brief

Liver Function Tests

MANY tests are available for the investigation of liver function, but most of them require the facilities offered by a well-equipped laboratory. This is always a limitation and one that undoubtedly has contributed to the disparity in information about liver function in man and domestic animals. Many investigations are essential in assessing any test, and in comparison with human medicine, there are relatively few routine facilities available to the veterinary clinician.

In addition there are basic biological factors common to both branches of medicine that complicate further the problems of liver function. These can be summarised by stating that the liver, in common with other organs, has a great variety of independent but sometimes interdependent activities, each with a wide normal range. Therefore, minor biochemical variations in some or any of these, due to any slight physiological or pathological cause, may not be recognised as definite departures from accepted normal values.

These observations lend support to the view that no liver function test should be performed in isolation, but in company with confirmatory tests and incorporated in the general clinical approach.

In general, there are three types of test. They are, first, investigation of blood flow through the liver by estimating clearance rates of artificially administered substances such as galactose and bromsulphalein (BSP), secondly, the determination of normal metabolites of, or associated with, the liver, *e.g.* serum globulin, alkaline phosphatase, cholinesterase, and transaminases, and thirdly, histochemical examination of isolated biopsy samples.

In an experimental study on African cattle, Garner (1952) applied many of these tests to normal animals and to animals dosed with carbon tetrachloride. Although trends in the variation of the tests were demonstrated, it is clear that most of the techniques are unsuitable for the demonstration of minor but possibly chronic dysfunctions of the liver.

It is encouraging to note that the development of new tests is improving the situation. Thus, at a recent symposium in America, Cornelius (1957) presented his experiences of liver function tests in small animals. Briefly, he suggested that a preliminary approach should include always determinations of icterus in blood, of bile pigments in urine, and an examination of faeces. As this is an over-simplified approach to a complex problem, particularly as icterus may not always be present, Cornelius suggested that other tests should be employed, notably BSP clearance and transaminase determination of serum proteins, because increased globulin may arise from causes other than liver dysfunction, notably, infectious diseases, nephrosis, starvation, etc. Alkaline phosphatase estimations are also somewhat unpredictable.

Probably the most promising approach is the determination of one or two of the transaminases found in blood. These are Serum (Plasma) Glutamic Oxaloacetic Transaminase-S(P)GOT, and Serum (Plasma) Glutamic Pyruvic Transaminase-S(P)GPT. The routine estimation of these substances for investigation of liver function was brought into prominence by Wróblewski and his colleagues (*e.g.* 1955, 1958), and they are now popular in many routine hospital laboratories.

At present they can be determined only in the laboratory, and as far as the author is aware no suitable modifications have been designed for use in the surgery or in routine practice. SGPT is formed mainly in the liver, and therefore damage to liver cells will release more of the enzymes that will be followed by increased blood levels. SGOT—a closely allied enzyme—is raised significantly in acute myocardial infarctions in the human subject, whereas SGPT is not, although both enzymes are increased in liver disease. Recent studies by O'Brien, Goble, and MacKay (1958) and by Pryse-Davies and Wilkinson (1958) suggest that in human subjects very high levels (more than 500 units) occur in infective hepatitis and in carbon tetrachloride poisoning, moderately high levels (100 to 300 units) in obstructive jaundice and in toxic hepatitis (excluding carbon tetrachloride poisoning), but variable and not very high levels in cirrhosis and in liver metastases. According to Pryse-Davies and Wilkinson (*loc. cit.*), the normal levels for SGOT and SGPT in man are up to 40 and 30 units respectively. In dogs the value of SGOT is about 50 units (Cornelius, *loc. cit.*). It is hoped that further information will be forthcoming about the normal and raised levels of these enzymes in domestic and farm animals, since this test appears to show great promise.

Another approach to the investigation of liver function in dogs has been made recently by Doggart, McCredie and Welbourn (1958) who investigated urinary amino-acids in normal and experimental animals by chromatographic analysis following artificially induced cirrhosis (carbon tetrachloride), portacaval anastomosis, and total arterialisation of the liver blood supply. The authors summarise the relation of liver function to urinary amino-acids succinctly, thus: "The amino-acid excretion in dogs with cirrhosis increased only when there was severe liver damage. When liver damage was less severe, there was selective amino-aciduria affecting taurine and glutamine." They further emphasise the important facts that there was little correlation between excretion of amino-acids and other liver function tests, and considerable variation in normal urinary amino-acid patterns.

These observations stress the need for applying any functional test, for liver or other organs, well within the general pattern of a full clinical investigation. All new and attractive analytical techniques in human and in veterinary medicine often go through a phase of over-emphasised popularity, the degree of which is measured by the initial success of tests.

(*Concluded in col. 2, page 617*)

The Victoria Veterinary Benevolent Fund

A general meeting of members of the Victoria Veterinary Benevolent Fund was held at 10, Red Lion Square, London, W.C.1, on June 11th.

There were present:—

Mr. E. Clark, Major J. J. Dunlop, Mr. R. C. U. Fisher, Mrs. D. I. Glover, Mr. H. A. Lamont, Professors J. McCunn, L. P. Pugh, Miss V. Tillemont-Thomason, Professor W. L. Weipers, Major W. H. Wortley.

Major J. J. Dunlop nominated Professor McCunn to take the chair and this was agreed.

The Secretary read the notice convening the meeting, as published in THE VETERINARY RECORD, and reported apologies for absence from Mr. E. P. Barrett, Captain W. Watt, and Captain A. Whicher.

Minutes

The Minutes of the previous general meeting, having been circulated and published in THE VETERINARY RECORD, were taken as read and signed as correct.

Resolution

Consideration was next given to the following resolution:—

"That the Articles of Association of the Association be altered as follows:—

Article 12. Substitute the words 'months of May or' for the words 'month of' in line 2 and delete the words 'on the day on which the Annual General Meeting of the Royal College of Veterinary Surgeons is held' in lines 3 and 4."

The Chairman said that at present the Fund must hold its annual general meeting in June on the same day as that fixed by the Royal College of Veterinary Surgeons for its annual general meeting. It was possible that in future the Royal College might be empowered to call its annual general meetings in May or in June. Therefore, for the Fund to be able to continue the present happy arrangement of holding its meeting on the same day, Article 12 must be altered.

The Council also felt that this opportunity should be taken to make provision for the Fund to have its annual general meeting on a date other than that on which the R.C.V.S. annual general meeting was to be held if circumstances made it necessary.

The Secretary said he had communicated with the Board of Trade and with the Registrar of the Joint Stock Companies, and the resolution as framed was in order.

The resolution was carried unanimously.

Election of President

Professor McCunn proposed the re-election of the President. This was seconded by Major Dunlop and carried unanimously.

Election of four Vice-Presidents

The Chairman said that on page 2 of the report there were listed Captain T. M. Mitchell, Professor L. P. Pugh, Mr. H. Sumner, and the late Professor G. H. Wooldridge. To their great regret they had not Professor Wooldridge with them any longer so it was necessary to appoint another Vice-President. They would notice there was no representative from the biggest civilian employer they had—the Ministry of Agriculture. He understood Mr. Dawes felt that it would be a very good thing if they had a Vice-President from the staff of the Ministry and he would like to propose from the chair that they asked Mr. Ritchie, the Chief Veterinary Officer, to honour them by being a Vice-President. This was seconded by Mr. H. A. Lamont and carried unanimously.

Major Dunlop was then unanimously re-elected hon. secretary, being proposed from the chair and seconded by Mr. D. I. Glover.

Professor McCunn was unanimously re-elected Honorary Treasurer, and the auditors, Messrs. Wilkinson, Chater & Co., were re-appointed.

Annual Report and Statement of Accounts

The annual report was considered page by page. Among matters raised was the Wortley Axe bequest of which the Chairman said: "The Council have been actively pursuing this matter for the last year or two. It has been constantly in their mind. It is being investigated now by Mr. Thatcher with the aid of Counsel to ascertain exactly what we can do and how we can do it. They have not reported fully yet, but the bequest will come up for discussion at the next meeting of the Wortley Axe committee which will be in the near future." Professor McCunn went on:—

"Before we leave pages 10 and 11, I think we must acknowledge what a wonderful effort our ladies make. The amount of income which flows into the fund through their efforts is remarkable. If it were not for them our annual income would not be very great, and we shall be ever grateful to them. I am sure you would wish me to place on record our gratitude to the ladies for all their efforts on behalf of the fund.

"Page 12 deals with the list of recipients. There are forty-seven cases, and I think this list shows the need for the fund. It also shows that the fund is doing extremely good work. There are many members of our profession and their dependants who would be in very dire straits if it were not for this fund and the generosity of those who subscribe to it. At the end of the list you will see that we paid out some £2,829 10s. 4d. You will also observe from the summary that 10 members, 25 widows, 1 wife, 10 daughters of late members, and 1 son of a late member were assisted."

Professor McCunn moved that the annual report and statement of accounts be received and adopted, and the motion was carried unanimously.

In Brief—*Concluded.*

If continued application gives successful results, it follows that the tests will take their place in normal routine procedures. This alone measures their worth as diagnostic tests in clinical medicine.

D. G. HARVEY, July 2nd, 1958.

References

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DOGGART, J. R., MCCREDIE, J. A., & WELBOURN, R. B. (1958). *Vet. Rec.* **70.** 279.

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KARMEN, A., WROBLEWSKI, F., & LADUE, J. S. (1955). *J. Clin. Invest.* **34.** 126.

O'BRIEN, E. N., GOBLE, A. J., & MACKAY, I. R. (1958). *Lancet.* **i.** 1,245.

PRYSE-DAVIES, J., & WILKINSON, J. H. (1958). *Ibid.* **i.** 1,249.

WROBLEWSKI, F., *et al.* (1957). "Hepatitis Frontiers" (Ford Foundation Symposia).

News and Comment

SPECIAL CHARTER FLIGHT TO THE ISLE OF MAN

Owing to the difficulty of reaching the Isle of Man on Sunday, the day immediately before the official opening of Congress, it has been suggested that a special aircraft might be chartered from London Airport. This would only be a practical possibility if a sufficient number of people wished to travel. Will anyone who would like to join such a flight please write *immediately* to the General Secretary of the B.V.A.? The possibility can then be fully explored. The single fare would be just over £6 10s.

R.C.V.S. EXAMINATIONS

Dublin

THIRD EXAMINATION

Hygiene only

Hand, P. J., Marray, E. A., and O'Driscoll, T. L.

Pathology only

Boothman, J. H., Boyce, W. T. M., Demery, V. W., Dickson, H. P., Duignan, J. J. A., McAuliffe, M. J., McParland, P. J., Phelan, N. P., and White, J. E. C.

FINAL EXAMINATION

Cousins, F. G. O., Duffy, E. F., Gaffney, W. M. N., Gormley, B. A., Harrington, D. P., Kelleher, D. L., Kenny, M. P., Leslie, N. W., Lyons, Valentine, McKinley, P. J., Moloney, J. B., Murray-Hayden, P. J., O'Brien, Patrick, O'Connell, John, O'Donnell, L. I., O'Neill, Alexander, O'Neill, Conal, Prendergast, M. J. G., Quill, T. S., Schlesinger, Percy, Sheahan, T. J., Smith, F. M., and Smyth, J. J.

London

THIRD EXAMINATION

Pathology only

Hassan, Y. M., Pandey, M. D. (credit), and Shettigara, P. T.

FINAL EXAMINATION

Ali, M. J., Angelo, S. J., Bhatia, T. R. S., Hussain, Iftikhar, May, J. N. T., Reid, P. A., Seneviratna, K. A. D. P. S., and Tripathy, G. S.

Cambridge

THIRD EXAMINATION

Pathology only

Gupta, B. N. (credit), and Sood, D. N.

FINAL EXAMINATION

Hervey, Miss Y. I.

Edinburgh

FINAL EXAMINATION

Dodson, G. T., and Maasri, K. J.

Glasgow

FINAL EXAMINATION

Carrigan, T. D., and Harrison, T. C.

THE PARLIAMENTARY AND SCIENTIFIC COMMITTEE

Dr. Wallace Brode, science adviser to the U.S. State Department, Washington, has been visiting this country from July 23rd to 26th. He kindly agreed to address a special meeting of the General Committee on Thursday, July 24th, at 5.30 p.m., in Committee Room 11, House of Commons. He spoke on International Science and other matters.

PERSONAL

Mr. Stewart Forrest, who was on the staff of the veterinary department, Birmingham, has now accepted a post as veterinary meat inspector to the County of Aberdeen Authority. A vacancy at Birmingham is advertised on another page.

Births

CLARKE.—On July 17th, at Crowborough Memorial Hospital, Sussex, to Barbara (*née* Cayless) and Peter B. Clarke, BS.V.SC., MS.R.C.V.S., a daughter (Philippa Mary), sister for Nicola Carolyn.

GOODHAND.—On July 16th, 1958, to Peggie, wife of R. H. Goodhand, B.SC., M.R.C.V.S., a son, Michael Ronald, brother for Keith.

JONES.—On July 6th, 1958, to Margaret, wife of Colin R. V. Jones, M.R.C.V.S., of 34, Trevethan Road, Falmouth, Cornwall, a daughter, Karen, sister for Ruth and Sarah.

RUTHERFORD.—On July 18th, 1958, at 20, Toppesfield Gardens, Hadleigh, Suffolk, to Theresa, wife of Leonard H. Rutherford, M.R.C.V.S., a daughter, Fiona.

Engagement

RUSSELL—TEBBUTT.—The engagement is announced between George Hugh Russell, M.R.C.V.S., youngest son of Mr. and Mrs. J. Kingsley Russell, of Hitchin, Herts, and Gillian Mary Tebbutt, elder daughter of Mr. and Mrs. Paul W. Tebbutt, also of Hitchin.

THE AGRICULTURAL EDUCATIONAL ASSOCIATION

At the yearly conference of this Association, which was held at the University of Reading on July 15th to 18th, Mr. G. N. Gould gave a paper on "Development of the Hampshire Cattle Breeders' Society, Artificial Insemination of Cattle and Pigs, and Progeny Testing."

The Society embraces a large membership derived from Principals and members of staff of farm institutes, professors and lecturers at agricultural colleges, and members of the N.A.A.S.

Mr. Gould addressed the Agricultural Section of the Conference, and there was a wide discussion after the reading of his paper. Mr. P. S. Marsden, Principal of the Lincolnshire (Lindsay) Farm Institute was in the chair.

SHIPLEY BOWLS CUP

The Eastern Counties Veterinary Society held their annual competition for the Shipley Bowls Cup at Stock on July 15th on the rinks of the Stock & Buttsbury Bowls Club. Sixteen members took part. Mr. C. H. Downe (Diss) won the cup for the third time, beating Dr. P. S. Pugh (Cambridge) in the final.

IN PARLIAMENT

Slaughterhouses (Meat Inspection)

SIR F. MEDLICOTT (July 17th) asked the Minister of Agriculture, Fisheries and Food if he is satisfied with the progress of the efforts to ensure that 100 per cent. of the meat passing through slaughterhouses is inspected; and if he will make a statement.

MR. GODBER: The replies to a questionnaire recently issued by my Ministry indicate that local authorities have made considerable progress during the last eighteen months, and that as much as 90 per cent. of the meat passing through slaughterhouses is now inspected. Local authorities are to be congratulated on this encouraging rate of progress, and my right hon. Friend is satisfied that they are doing their best to maintain this progress and to raise the standard of inspection despite the great pressure on inspecting staff in many areas.

DR. STROSS: Would the Minister also take steps to see that there is *ante-mortem* inspection of cattle? Is he aware that *post-mortem* inspection of meat alone is not sufficient to protect the public health?

MR. GODBER: I am aware of the hon. Member's feelings about this. We discussed the subject at some length during the current session. At the moment I think we must concentrate on *post-mortem* inspection, but I do not set that as an end to all inspection. I realise the importance of what he said.

Irish Cattle (Import)

MR. J. JOHNSON (July 17th) asked the Minister if he is aware of the anxiety of Warwickshire farmers regarding the existing arrangements for the importation of Irish cattle; whether he is yet satisfied with the standard of Irish attestation; and if he will make a further statement on this matter.

MR. GODBER: Pending the eradication of bovine tuberculosis from Great Britain the position is adequately safeguarded by the requirement that once-tested cattle must be isolated for a period and pass a second test before being added to attested herds. My right hon. Friend would, however, welcome the early development of a trade in attested Irish cattle. The future of the trade in Irish cattle after eradication of the disease from Great Britain is under active discussion between the authorities concerned. My right hon. Friend is aware of the concern of farmers with these matters, but he is not in a position to make a further statement at present.

MR. JOHNSON: In view of the amounts that the Government spend to ensure that our own cattle are fully attested, is not it foolish to continue importing cattle which have been only once attested before coming here? Would not it be desirable to have arrangements on the Irish side comparable with those that we have here?

MR. GODBER: I entirely agree with the hon. Member. We want the Irish to speed up this matter as much as possible. We have been in negotiation with them on a number of occasions. I myself have discussed it with some of their representatives. We want the situation improved as soon as possible, but at the moment the position is safeguarded in the way indicated in my original Answer.

UNIVERSITY NEWS

Bristol

A very distinguished company, including the Pro-Chancellor, the Chairman of Council, the vice-Chancellor, the Lord Mayor, and the President of the R.C.V.S. was present at the University Veterinary Laboratories, Langford, on July 11th, when the new surgical wing was officially declared open. The ceremony was performed by Sir Wilfrid Anson who presented the key of the new building to Professor Messervy who holds the Chair of Surgery in the Veterinary School. A full account of the ceremony, with photographs, will be published next week.

FIFTY YEARS AGO

The following extract is taken from a Presidential address to the Western Counties V.M.A.:—

"Some of the dispensing by veterinary surgeons is, I know, absolutely disgraceful. Draughts sent out in ginger beer bottles, powders wrapped up in newspaper etc., labels competing in design with those on a special brand of mustard or pickles. I have seen labels embellished with a horse 'rampant' surrounded by a galaxy of medals.

"It is coming to something when insurance companies offer to supply members of the profession with labels, conditionally that the company's advertisement is printed thereon. Some of the account forms are not much better, particularly when the practitioner's name is embellished with the mystic letters.

"A short time ago a town practitioner told me that if ever he should be in the country and see a man driving an evil-smelling trap, with a stick with a loop of string tied to it sticking out behind, he generally finds on enquiry that the driver is the local veterinary surgeon. I will not comment on this.

"We know to our great regret that there are men who are anything but ornaments to the profession, men whose actions are questionable, the places they frequent and the company they keep more so, but this class of man is dying out, thanks in a great measure to the high standard of the entrance examination, and to the affiliation of some of our teaching schools to the universities.

"We cannot all keep up large establishments with a big staff of assistants and servants, but each individual can try to look beyond his day book and endeavour to advance the interest of the profession by adding to the scientific and clinical knowledge. There are brilliant examples we can follow, and we ought to be proud that we can claim such men as Sir John M'Fadyean, Mr. William Hunting, and others too numerous to mention. We can all be gentlemen. If we only respect our calling, the public will respect us."

R.C.V.S. OBITUARY

We record with regret the death of the following member of the profession:—

LITTLER, Lionel, Elgin Lodge, Scalford Road, Melton Mowbray, Leics. Graduated December 18th, 1914, at Edinburgh. Died July 18th, 1958, aged 67.

COMING EVENTS

July

29th (Tues.). Dinner Meeting of Metropolitan Region, B.S.A.V.A., at Royal Society of Medicine, at 7 p.m.

September

17th (Wed.). Meeting of the North Wales Division at Glynllifon, Caernarvon.

21st to 26th (Official Opening, Monday, September 22nd). 76th Annual Congress of the British Veterinary Association in Douglas, Isle of Man.

ADDRESSES OF DISEASE INFECTED PREMISES

The list given below indicates, first the county in which are situated the premises on which disease has been confirmed, followed by the postal address and date of outbreak.

Anthrax

Lanarks. West Hookhead Farm, Strathaven (July 16).
Oxford. Glen Farm, Warmington, Banbury (July 17).

Fowl Pest

Cambs. 15, Church Street, March; 4, Jobs Lane, March; 108, High Street, Chatteris; Nightlayer, Chatteris (July 14); Emlynian, Knights End, March; The Limes, Knights End, March (July 16).

Norfolk. Glebe Land, Roughton, Norwich (July 12); Northcote, Bunweil, Norwich (July 15); Lilac Farm, Hevingham, Norwich (July 17).

Sussex. Lower Est Street Farm, Maynards Green, Horam (July 11).

Swine Fever

Beds. Park Farm, Bletsoe (July 16).
Cambs. Hole Farm, Witchford Road, Ely (July 15).
Devon. Lower Bagmore Farm, Exton, Exeter (July 11).
Dorset. Ivy House Farm, Oborne, Sherborne (July 14); Orchard Farm, Stalbridge, Sturminster, Newton (July 16).
Essex. Priestfields Farm, Castle Hedingham (July 11).
Herts. Hunsdon House Farm, Hundson, Ware (July 14).
Lancs. Farnworth Close Farm, Church Street, Westhoughton; Higher Broadfield Farm, Heywood (July 11); Wicken Bank Farm, Manchester Road, Heywood; Walsh Fold Farm, Turton, Bolton (July 15).
Norfolk. Bungalow Farm, Gimingham, Norwich (July 11); Oak Farm, Poringland, Norwich (July 14); Park Farm, Deopham, Wymondham (July 16).
Somerset. Rectors Farm, Rectors Way, Weston-super-Mare (July 14).
Suffolk. Winton, Gt. Bealings, Woodbridge (July 17).
Warwicks. Manor Farm, Ryton-on-Dunsmore, Coventry (July 15); Ballards Green Farm, Arley, Coventry (July 17).
Wilts. Park Copse Farm, Hook, Wootton Bassett, Swindon (July 14); Marlborough House, Marlborough Road, Wroughton, Swindon; Piggeries, Wharf Road, Wroughton, Swindon; Lynwood, Devizes Road, Bromham, Chippenham (July 15).
Worcs. Dunclent Farm, Stone, Kidderminster (July 15).

Letters to the Editor

The views expressed in letters addressed to the Editor represent the personal opinions of the writer only and their publication does not imply endorsement by the B.V.A.

"Doctor" or "Mister"?

Sir,—The annual recrudescence in correspondence over our title, seems to show that some are suffering from an inferiority complex, and desire to cloak their occupation by a title which by no means is now confined to those expected to heal the sick, whether human or animal.

Personal contacts with members of the medical profession seem clearly to show that the surgeon expects to be addressed as "Mister" and is inclined to correct one if called "Doctor." The verb "to doctor" may be used to describe forms of commercial or other faking: there can be no mistake what the verb "to vet" implies, when applied to animal, vegetable, or mineral.

The status of a person, if this is the real issue at stake, is determined by his behaviour and ability to give the services expected. No amount of camouflage will assist, if these are absent.

Yours faithfully,
J. F. D. TUTT.

"Rothiemurchus,"
Winchester.
July 16th, 1958.

Hysterotomy to Obtain Disease-free Pigs

Sir,—To substantiate the claims made for this method of obtaining disease-free pigs and to establish a comparison between American and British results, I would be grateful to members of the veterinary profession who can help me with information on the following points:—

1. Approximate immediate costs of aseptic farrowing.
2. Representative data of conversion ratio, grading, age at slaughter, costs, etc., before and after aseptic farrowing in a herd.
3. The reason for undertaking aseptic farrowing, and the mode of sow-selection for aseptic farrowing (*i.e.* scope of records, etc.).
4. The diet on which aseptically farrowed pigs were raised, growth rate, etc.
5. Occurrence of breakdowns after aseptic farrowing, and the causes.
6. Any other relevant observations and photographic material would be greatly appreciated.

All correspondence will, of course, be treated as confidential.

Yours faithfully,
F. VOGT.

North Tuddenham,
Norfolk.
July 17th, 1958.

Erratum

In the letter published last week over the signatures of A. W. J. Broome and G. E. Lamming, *Eimeria coli* should have read *Escherichia coli*.